

National  
Library  
of MedicineMy NC  
[Sign In] [Regis]

All Databases

PubMed

Nucleotide

Protein

Genome

Structure

OMIM

PMC

Journals

Book

Search PubMed



for

Go

Clear

Limits

Preview/Index

History

Clipboard

Details

Display

Abstract



Show

20



Sort by



Send to



About Entrez

Text Version

All: 1

Review: 0



Entrez PubMed

Overview

Help | FAQ

Tutorial

New/Noteworthy

E-Utilities

PubMed Services

Journals Database

MeSH Database

Single Citation Matcher

Batch Citation Matcher

Clinical Queries

LinkOut

My NCBI (Cubby)

Related Resources

Order Documents

NLM Catalog

NLM Gateway

TOXNET

Consumer Health

Clinical Alerts

ClinicalTrials.gov

PubMed Central

☐ 1: Proc Natl Acad Sci U S A. 1992 Dec 15;89(24):11949-53. Related Articles, LinksFREE full text article at  
www.pnas.orgFREE full text article  
in PubMed Central**Detection of glutamine synthetase in the cerebrospinal fluid of Alzheimer diseased patients: a potential diagnostic biochemical marker.****Gunnersen D, Haley B.**

Department of Biochemistry, College of Pharmacy, University of Kentucky, Lexington 40536-0084.

In this report, 8- and 2-azidoadenosine 5'-[gamma-32P]triphosphate were used to examine cerebrospinal fluid (CSF) samples for the presence of an ATP binding protein unique to individuals with Alzheimer disease (AD). A 42-kDa ATP binding protein was found in the CSF of AD patients that is not observed in CSF from normal patients or other neurological controls. The photolabeling is saturated with 30 microM 2-azidoadenosine 5'-[gamma-32P]triphosphate. Photoinsertion can be totally prevented by the addition of 25 microM ATP. Photoinsertion of 2-azidoadenosine 5'-triphosphate into the protein is only weakly protected by other nucleotides such as ADP and GTP, indicating that this is a specific ATP binding protein. A total of 83 CSF samples were examined in a blind manner. The 42-kDa protein was detected in 38 of 39 AD CSF samples and in only 1 of 44 control samples. This protein was identified as glutamine synthetase [GS; glutamate-ammonia ligase; L-glutamate:ammonia ligase (ADP-forming), EC 6.3.1.2] based on similar nucleotide binding properties, comigration on two-dimensional gels, reaction with a polyclonal anti-GS antibody, and the presence of significant GS enzyme activity in AD CSF. In brain, GS plays a key role in elimination of free ammonia and also converts the neurotransmitter and excitotoxic amino acid glutamate to glutamine, which is not neurotoxic. The involvement of GS, if any, in the onset of AD is unknown. However, the presence of GS in the CSF of terminal AD patients suggests that this enzyme may be a useful diagnostic marker and that further study is warranted to determine any possible role for glutamate metabolism in the pathology of AD.

PMID: 1361232 [PubMed - indexed for MEDLINE]

---

Display  Show  Sort by  Send to

[Write to the Help Desk](#)  
[NCBI](#) | [NLM](#) | [NIH](#)  
Department of Health & Human Services  
[Privacy Statement](#) | [Freedom of Information Act](#) | [Disclaimer](#)

Mar 29 2005 17:30:14